Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Table: Ovarian cortex concentration of PDF, PRF, SEF, TRF, AMH, Ki67, and Caspase3, in the four groups.

Variable	Baseline Median (Q1, Q3)	Placebo Group Median (Q1, Q3)	rAMH Group Median (Q1, Q3)	AMHR2BP Group Median (Q1, Q3)	p-value
PDF/mm^3	1195 (1191, 1248)	614 (538, 705)	1182 (1095, 1248)	1012 (916, 1134)	0.011
PRF/mm^3	788 (670, 817)	1165 (1152, 1176)	514 (497, 548)	383 (306, 533)	0.016
SEF/mm^3	970 (968, 1061)	1613 (1528, 1664)	1144 (1119, 1300)	1371 (852, 1662)	ns
TEF/mm^3	583 (579, 606)	1082 (1076, 1119)	871 (808, 890)	587 (519, 707)	0.045
Inhibin B (pg/μg RNA)	34.53 (34.53, 34.54)	48.41 (47.36, 49.47)	4.07 (3.86, 4.27)	2.22 (1.83, 2.60)	0.001
Ki67 (pg/μg RNA)	22.84 (20.49, 25.21)	69.26 (67.40, 71.12)	8.05 (7.20, 8.89)	5.90 (5.77, 6.02)	0.005
Caspase3 (pg/μg RNA)	1.31 (1.27, 1.36)	3.10 (3.00, 3.20)	0.46 (0.43, 0.50)	0.32 (0.31, 0.33)	0.004

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HUMAN PRE-IMPLANTATION EMBRYOS ARE PERMISSIVE TO SARS-COV-2 ENTRY.

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OBJECTIVE: To determine whether human pre-implantation embryos have the potential to be infected by SARS-CoV-2, the virus responsible for COVID-19.

DESIGN: Assessment of expression levels of SARS-CoV-2 entry mediators in human embryo biopsies by RNAseq analysis, and infection of cultured embryos with SARS-CoV-2 Spike glycoprotein pseudotyped reporter virions expressing green fluorescent protein (GFP).

MATERIALS AND METHODS: Trophectoderm biopsies from blastocyst-stage embryos (n=24) were processed for RNAseq using a commercial kit and sequenced; results were analyzed for expression of factors implicated in SARS-CoV-2 cellular entry. For viral infection experiments, blastocyst-stage embryos (n=94) were hatched from zonas mechanically, and infected by spinoculation with GFP-reporter virions pseudotyped with the SARS-CoV-2 Spike glycoprotein (required for SARS-CoV-2 entry). Embryos were subsequently monitored for fluorescence at 24-48 hours post-infection. Various control conditions were used as specified in the 'results' section. A mixed population of euploid, aneuploid, and untested embryos used in the study were from IVF treatment, donated to research by signed informed consent. The project was approved by an Institutional Review Board.

RESULTS: Cells collected from blastocyst-stage embryos robustly expressed the canonical SARS-CoV-2 entry receptor *ACE2* and the putative activator protease *TMPRSS2*, in addition to other reported entry factors. Embryos exposed to reporter virions pseudotyped with SARS-CoV-2 Spike glycoprotein displayed robust GFP signal, often in numerous cells with cytoplasmic localization. Specificity was confirmed by the absences of fluorescence in embryos treated with virions lacking the Spike glycoprotein ("bald" virus), or when embryos were spinoculated with media alone in the absence of virus. Embryos exposed to Spike glycoprotein-positive reporter virus in the presence of neutralizing anti Spike- or anti-ACE2-blocking antibodies exhibited negligible GFP signal, while control monoclonal IgG antibody-treated embryos maintained GFP expression. These results implicated the canonical Spike-ACE2 axis in the viral entry. Lastly, embryos exposed to reporter virions pseudotyped with Spike glycoprotein of SARS-CoV-1 (which also enters cells via ACE2) displayed GFP fluorescence, while embryos exposed to reporter viruses pseudotyped with Spike glycoprotein of MERS (which utilizes Dipeptidyl Peptidase IV (DPP4) instead of ACE2) resulted in no fluorescence.

CONCLUSIONS: Our results indicate that cells present in pre-implantation embryos are permissive to the canonical Spike-ACE2 viral entry mechanism utilized by SARS-CoV-2. These results encourage further investigation into the potential of SARS-CoV-2 infection in human embryos and may have wider implications in natural conception and ART practice.